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Commentary

Cellular Immunity in Neuroinflammatory Disorders: Mechanisms and Therapies

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DESCRIPTION

Neuroinflammatory disorders, encompassing conditions such as Multiple Sclerosis (MS), Neuromyelitis Optica Spectrum Disorder (NMOSD), and certain forms of autoimmune encephalitis, represent a complex interplay between the Central Nervous System (CNS) and the immune system. Among the myriad components of immunity, cellular immunity mediated primarily by T lymphocytes, B lymphocytes, natural killer cells, and microglia plays a pivotal role in both the initiation and progression of neuroinflammation. Understanding these cellular mechanisms is not only critical for elucidating disease pathogenesis but also for guiding the development of innovative therapeutic strategies that can effectively modulate immune responses while minimizing collateral CNS damage.

Mechanisms of cellular immunity in neuroinflammation

The CNS has traditionally been considered an immune-privileged site due to the presence of the Blood-Brain Barrier (BBB), that restricts the entry of circulating immune cells. However, in neuroinflammatory disorders, this barrier is often disrupted, allowing immune cells to infiltrate the CNS and trigger inflammatory cascades. T cells, particularly helper T cells, are central to this process. In multiple sclerosis, for instance, autoreactive T cells recognize myelin antigens and initiate a cascade of inflammatory responses that lead to demyelination and neuronal injury. Th1 cells contribute through the release of pro-inflammatory cytokines such as interferon-gamma and tumor necrosis factor-alpha, while Th17 cells exacerbate inflammation interleukin-17, promoting BBB breakdown and recruitment of additional immune cells.

B cells also contribute to neuroinflammation, not only through antibody production but also by functioning as Antigen-Presenting Cells (APCs) and cytokine producers. In conditions like NMOSD, pathogenic autoantibodies targeting aquaporin-4 are produced by B cells and plasma cells, leading to complement-mediated cytotoxicity and astrocyte damage. Beyond their classical roles, B cells secrete cytokines such as interleukin-6 and granulocyte-macrophage colony-stimulating factor, that further modulate T cell responses and amplify CNS inflammation.

Microglia, the resident immune cells of the CNS, act as both sensors and effectors of neuroinflammation. In healthy conditions, microglia maintain homeostasis by clearing cellular debris and regulating synaptic plasticity. During neuroinflammatory states, however, they adopt an activated phenotype, releasing pro-inflammatory mediators. This microglial activation contributes to neuronal damage, oligodendrocyte loss, and demyelination, thereby amplifying the pathology initiated by infiltrating lymphocytes.

Moreover, recent research highlights the role of regulatory immune cells in controlling neuroinflammation. Regulatory T cells (Tregs) and certain subsets of B cells can mitigate tissue damage by producing anti-inflammatory cytokines such as interleukin-10 and transforming growth factor-beta. Dysregulation or depletion of these regulatory populations often correlates with disease severity, suggesting that restoring the balance between pro-inflammatory and anti-inflammatory cellular responses could be therapeutically beneficial.

Therapeutic implications of cellular immunity in neuroinflammatory disorders

Insights into cellular immunity have revolutionized therapeutic approaches for neuroinflammatory disorders. Traditional therapies, such as corticosteroids and immunosuppressants, broadly dampen immune responses but are associated with significant side effects and limited long-term efficacy. Modern strategies aim to target specific immune cell populations or signaling pathways involved in disease pathology, offering more precise and potentially safer interventions.

Beyond cell depletion, modulating immune cell function has gained attention. Therapies aimed at enhancing regulatory T cell activity or inducing tolerogenic B cells could restore immune homeostasis without completely suppressing protective immune functions. Additionally, small molecule inhibitors targeting intracellular signaling pathways, can selectively inhibit T cell

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activation and trafficking, offering alternative approaches to conventional immunosuppression.

Another promising area is the modulation of microglial activation. Preclinical studies suggest that targeting microglial signaling pathways, such as the *NLRP3* inflammasome or colony-stimulating factor 1 receptor (CSF1R), can reduce neuroinflammation and neuronal damage. Such strategies hold the potential to not only limit immune-mediated injury but also promote tissue repair and functional recovery.

Finally, combination therapies are increasingly recognized as essential for managing complex neuroinflammatory disorders. By simultaneously targeting multiple immune cell types or pathways, combination approaches can achieve synergistic effects, reduce disease activity, and potentially delay progression while minimizing adverse effects.

CONCLUSION

Cellular immunity is at the heart of neuroinflammatory disorders, orchestrating both protective and pathogenic responses within the CNS. T cells, B cells, microglia, and regulatory immune populations interact in intricate networks that determine disease onset, progression, and severity. Advances in understanding these mechanisms have driven the development of targeted therapies that modulate specific immune components, offering greater efficacy and safety compared with traditional broad-spectrum immunosuppression. As research continues to unravel the cellular and molecular underpinnings of neuroinflammation, the prospect of personalized immunotherapy tailored to the unique immune profile of each patient becomes increasingly attainable. The continued integration of mechanistic insights with innovative therapeutic strategies promises a future in that neuroinflammatory disorders can be more effectively controlled, and patient outcomes significantly improved.